Stellingen

Cerebral Neurotrophins and Behavioral Aspects of a Neurodevelopmental Model of Schizophrenia

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1. The maldevelopmental model of schizophrenia postulates pathological alterations in embryonal neurogenesis involving several brain areas as the etiopathogenetic basis of schizophrenic psychosis; the neurotrophic factor hypothesis explains these changes as the result of disturbances of processes involving the trophic factors (J Neural Transm. 1998-105:85-100).

2. Effects during brain development lead to defective neural connectivity and altered biochemical functioning resulting in any dysfunction later in life, including cognition, emotions and attentional abilities.

3. Different kinds of acute and chronic stress induce changes in the content of neurotrophins in the brain.

4. Schizophrenics show alteration in the levels of neurotrophic factors in both brain and serum.

5. Social behavior and learning and memory processes are finely controlled by endogenous neurotrophins' presence.

6. The reliability of an animal model depends on the comparability with aspects of some human processes, the limit that should be taken into consideration is that animals are not humans.

7. The difference between science and opinion is that science begets knowledge whereas opinion generates ignorance (Hippocrates).

8. The primary lesion(s) in schizophrenia does not necessarily involve dopamine, glutamate, and GABA directly but could deal with a more general defect, such as a faulty connectivity of developmental origin (Annu Rev Pharmacol Toxicol. 2001-41:237-260).

9. Therapeutic efficacy of drugs blocking dopamine receptors does not prove that cerebral dopamine-containing neurons function abnormally in schizophrenia (Arch Gen Psychiatry. 2003 60:974-977).

10. The schizophrenia model proposed by Lipska and coworkers has no full heuristic values because it is based on the assumption of a discrete and single brain lesion being the main cause of the disorder (Neuropsychopharmacology. 1993-9:67-75).

11. Gene polymorphism of neurotrophins are not and cannot be associated with the vulnerability to develop schizophrenia since the brain damage has already occurred so the recovery of the neuronal circuitry is hard to carry out.